

WEST Search History

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DATE: Tuesday, July 06, 2004

Hide?	Set Name	Query	Hit Count
<i>DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=AND</i>			
<input type="checkbox"/>	L1	invaplex	9

END OF SEARCH HISTORY

[Generate Collection](#)[Print](#)**Search Results - Record(s) 1 through 9 of 9 returned.**

- 
- ☐ 1. [20020197276](#). 17 May 02. 26 Dec 02. Heterologous protection induced by immunization with invaplex vaccine. Oaks, Edwin V., et al. 424/203.1; A61K039/116.
- 
- ☐ 2. [20010009957](#). 31 Jan 01. 26 Jul 01. Invaplex from gram negative bacteria, method of purification and methods of use. Oaks, Edwin W., et al. 530/395; 435/195 C12N009/14 C07K014/24.
- 
- ☐ 3. [6680374](#). 31 Jan 01; 20 Jan 04. Invaplex from gram negative bacteria, method of purification and methods of use. Oaks; Edwin V., et al. 530/388.1; 424/130.1 424/141.1 424/150.1 424/164.1 435/329 435/332 435/340 530/350 530/388.2 530/388.4. C07K016/00 C12P021/08.
- 
- ☐ 4. [6277379](#). 29 Sep 99; 21 Aug 01. Use of purified invaplex from gram negative bacteria as a vaccine. Oaks; Edwin V., et al. 424/197.11; 424/193.1 424/203.1 424/234.1 424/241.1 424/249.1 424/252.1 424/258.1 435/975 530/350 536/123.1. A61K039/385.
- 
- ☐ 5. [6245892](#). 29 Sep 99; 12 Jun 01. Invaplex from gram negative bacteria, method of purification and methods of use. Oaks; Edwin V., et al. 530/350; 424/282.1 435/7.2 530/416. C07K014/00 C07K001/00 G01N033/53 A61K045/00.
- 
- ☐ 6. [WO002094190A2](#). 17 May 02. 28 Nov 02. HETEROLOGOUS PROTECTION INDUCED BY IMMUNIZATION WITH INVAPLEX VACCINE. OAKS, EDWIN V, et al. A61K00/;.
- 
- ☐ 7. [WO 200294190A](#). Inducing in a subject a protective immune response against infection with a first invasive gram-negative bacteria by administering a composition comprising Invaplex 50 from a second heterologous invasive gram negative bacteria. OAKS, E V, et al. A61K000/00 A61K039/02 A61K039/108 A61K039/112 A61K039/116 A61K039/385 C07K001/00 G01N033/53.
- 
- ☐ 8. [US 6277379B](#). New vaccine for protection against infection with gram-negative bacteria comprising Invaplex of the bacteria to elicit protective antibodies. HARTMAN, A B, et al. A61K000/00 A61K039/02 A61K039/385 A61P031/04.
- 
- ☐ 9. [US 6245892B](#). New composition comprising isolated Invaplex of gram-negative bacteria comprising at least one invasin protein associated with LPS of the gram-negative bacteria. OAKS, E W, et al. A61K000/00 A61K045/00 C07K001/00 C07K014/00 C07K014/24 C07K016/00 C12N009/14 C12P021/08 G01N033/53.
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**Search Results - Record(s) 1 through 9 of 9 returned.**

- ☐ 1. 20020197276. 17 May 02. 26 Dec 02. Heterologous protection induced by immunization with invaplex vaccine. Oaks, Edwin V., et al. 424/203.1; A61K039/116.
- ☐ 2. 20010009957. 31 Jan 01. 26 Jul 01. Invaplex from gram negative bacteria, method of purification and methods of use. Oaks, Edwin W., et al. 530/395; 435/195 C12N009/14 C07K014/24.
- ☐ 3. 6680374. 31 Jan 01; 20 Jan 04. Invaplex from gram negative bacteria, method of purification and methods of use. Oaks; Edwin V., et al. 530/388.1; 424/130.1 424/141.1 424/150.1 424/164.1 435/329 435/332 435/340 530/350 530/388.2 530/388.4. C07K016/00 C12P021/08.
- ☐ 4. 6277379. 29 Sep 99; 21 Aug 01. Use of purified invaplex from gram negative bacteria as a vaccine. Oaks; Edwin V., et al. 424/197.11; 424/193.1 424/203.1 424/234.1 424/241.1 424/249.1 424/252.1 424/258.1 435/975 530/350 536/123.1. A61K039/385.
- ☐ 5. 6245892. 29 Sep 99; 12 Jun 01. Invaplex from gram negative bacteria, method of purification and methods of use. Oaks; Edwin V., et al. 530/350; 424/282.1 435/7.2 530/416. C07K014/00 C07K001/00 G01N033/53 A61K045/00.
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- ☐ 7. WO 200294190A. Inducing in a subject a protective immune response against infection with a first invasive gram-negative bacteria by administering a composition comprising Invaplex 50 from a second heterologous invasive gram negative bacteria. OAKS, E V, et al. A61K000/00 A61K039/02 A61K039/108 A61K039/112 A61K039/116 A61K039/385 C07K001/00 G01N033/53.
- ☐ 8. US 6277379B. New vaccine for protection against infection with gram-negative bacteria comprising Invaplex of the bacteria to elicit protective antibodies. HARTMAN, A B, et al. A61K000/00 A61K039/02 A61K039/385 A61P031/04.
- ☐ 9. US 6245892B. New composition comprising isolated Invaplex of gram-negative bacteria comprising at least one invasin protein associated with LPS of the gram-negative bacteria. OAKS, E W, et al. A61K000/00 A61K045/00 C07K001/00 C07K014/00 C07K014/24 C07K016/00 C12N009/14 C12P021/08 G01N033/53.

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Terms	Documents
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File 155:MEDLINE(R) 1951-2004/Sep W2

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\*File 155: Medline has been reloaded. Accession numbers  
have changed. Please see HELP NEWS 154 for details.

Set Items Description

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Cost is in DialUnits

?ds

Set	Items	Description
S1	139	E3-E5
S2	121	E3-E4
S3	12	'IPAD PROTEIN, SHIGELLA FLEXNERI'
S4	94	'IPAD'
S5	223	'VIRG PROTEIN' OR 'VIRG'
S6	28536	LPS
S7	1	'IPALS'
S8	44	'IPAC PROTEIN'
S9	427	S1 OR S2 OR S3 OR S4 OR S5 OR S8
S10	1943	S6 AND (SHIG? OR ESCHER? OR SALMON? OR YERSIN? OR RICKETT? OR BRUCELL? OR ERHLICH? OR CAMPYLOBACT? OR LEGIONELL? OR NEIS- SER? OR EDWARDSI?)/TI
S11	12678	LIPOPOLYSAC?/TI OR LPS?/TI
S12	965	S10 AND S11

?s s9/1998:2004

	427	S9
	3378725	PY=1998 : PY=2004
S13	155	S9/1998:2004

?s s12/1998:2004

	965	S12
	3378725	PY=1998 : PY=2004
S14	322	S12/1998:2004

?ds

Set	Items	Description
S1	139	E3-E5
S2	121	E3-E4
S3	12	'IPAD PROTEIN, SHIGELLA FLEXNERI'
S4	94	'IPAD'
S5	223	'VIRG PROTEIN' OR 'VIRG'
S6	28536	LPS
S7	1	'IPALS'
S8	44	'IPAC PROTEIN'
S9	427	S1 OR S2 OR S3 OR S4 OR S5 OR S8
S10	1943	S6 AND (SHIG? OR ESCHER? OR SALMON? OR YERSIN? OR RICKETT? OR BRUCELL? OR ERHLICH? OR CAMPYLOBACT? OR LEGIONELL? OR NEIS- SER? OR EDWARDSI?)/TI
S11	12678	LIPOPOLYSAC?/TI OR LPS?/TI
S12	965	S10 AND S11
S13	155	S9/1998:2004
S14	322	S12/1998:2004

?s s9 not s13

	427	S9
	155	S13
S15	272	S9 NOT S13

?s s12 not s14

	965	S12
	322	S14
S16	643	S12 NOT S14

?s s15 and (gram near5 negativ?)

	272	S15
	0	GRAM NEAR5 NEGATIV?
S17	0	S15 AND (GRAM NEAR5 NEGATIV?)

?s s15 and (gram (5n) negativ?)

	272	S15
	58917	GRAM

389371 NEGATIV?  
 37151 GRAM(5N)NEGATIV?  
 S18 7 S15 AND (GRAM (5N) NEGATIV?)  
 ?s s15 and (SHIG? OR ESCHER? OR SALMON? OR YERSIN? OR RICKETT? OR BRUCELL? OR ERHLICH?  
 OR CAMPYLOBACT? OR LEGIONELL? OR NEISSER? OR EDWARDSI?)  
 272 S15  
 14181 SHIG?  
 216986 ESCHER?  
 66052 SALMON?  
 8563 YERSIN?  
 7108 RICKETT?  
 11766 BRUCELL?  
 26 ERHLICH?  
 8805 CAMPYLOBACT?  
 4773 LEGIONELL?  
 16355 NEISSER?  
 367 EDWARDSI?  
 S19 174 S15 AND (SHIG? OR ESCHER? OR SALMON? OR YERSIN? OR  
 RICKETT? OR BRUCELL? OR ERHLICH? OR CAMPYLOBACT? OR  
 LEGIONELL? OR NEISSER? OR EDWARDSI?)  
 ?s s19 and s16  
 174 S19  
 643 S16  
 S20 1 S19 AND S16  
 ?t s20/9/all

20/9/1

DIALOG(R) File 155:MEDLINE(R)

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10042897 PMID: 8157605

**Nucleotide sequence of the rhamnose biosynthetic operon of *Shigella flexneri* 2a and role of lipopolysaccharide in virulence.**

Rajakumar K; Jost B H; Sasakawa C; Okada N; Yoshikawa M; Adler B  
 Department of Microbiology, Monash University, Clayton, Victoria,  
 Australia.

Journal of bacteriology (UNITED STATES) Apr 1994, 176 (8) p2362-73,  
 ISSN 0021-9193 Journal Code: 2985120R

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

N1308, a chromosomal Tn5 mutant of *Shigella flexneri* 2a, was described previously as a lipopolysaccharide ( LPS ) mutant with a short O side chain. N1308 formed foci, but not plaques, in LLC-MK2 cell monolayers and was negative in the Sereny test. In this study, the wild-type locus inactivated in N1308 was cloned and further defined by means of complementation analysis. A 4.3-kb BstEII-XhoI fragment of *S. flexneri* 2a YSH6200 DNA was sufficient to restore both normal LPS and virulence phenotype to the mutant. DNA sequencing of this region revealed four genes, rfbA, rfbB, rfbC, and rfbD, encoding the enzymes required for the biosynthesis of activated rhamnose. The four genes were expressed in *Escherichia coli*, and the expected protein products were visualized by sodium dodecyl sulfate-polyacrylamide gel electrophoresis. N1308 was shown to have normal levels of surface IpaC and IpaD, while a Western blot (immunoblot) of whole-cell lysates or outer membrane fractions indicated an elevated level of appropriately localized VirG. An in vitro invasion assay revealed that N1308 had normal primary invasive capacity and was able to multiply and move normally within the initial infected cell. However, it exhibited a significant reduction in its ability to spread from cell to cell in the monolayer. A double immunofluorescence assay revealed differences between LLC-MK2 cells infected with the wild-type YSH6000 and those infected with N1308. The wild-type bacteria elicited the formation of the characteristic F-actin tails, whereas N1308 failed to do so. However, N1308 was capable of inducing deposition of F-actin, which accumulated in a peribacterial fashion with only slight, if any, unipolar accumulation of

the cytoskeletal protein.

Tags: Support, Non-U.S. Gov't

Descriptors: Bacterial Proteins--genetics--GE; \*Genes, Bacterial  
--physiology--PH; \*Lipopolysaccharides; \*Rhamnose--genetics--GE; \* *Shigella*  
*flexneri*--genetics--GE; \* *Shigella* *flexneri*--pathogenicity--PY; Amino  
Acid Sequence; Bacterial Proteins--physiology--PH; Base Sequence; Cloning,  
Molecular; DNA Restriction Enzymes; DNA Transposable Elements--genetics--GE  
; Genetic Complementation Test; Molecular Sequence Data; Mutation  
--physiology--PH; Operon--genetics--GE; Operon--physiology--PH; Rhamnose  
--biosynthesis--BI; Virulence--genetics--GE; Virulence--physiology--PH

Molecular Sequence Databank No.: GENBANK/L14842

CAS Registry No.: 0 (Bacterial Proteins); 0 (DNA Transposable  
Elements); 0 (Lipopolysaccharides); 10485-94-6 (Rhamnose)

Enzyme No.: EC 3.1.21 (DNA Restriction Enzymes)

Gene Symbol: rfb

Record Date Created: 19940513

Record Date Completed: 19940513

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S2	121	E3-E4
S3	12	'IPAD PROTEIN, SHIGELLA FLEXNERI'
S4	94	'IPAD'
S5	223	'VIRG PROTEIN' OR 'VIRG'
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S7	1	'IPALS'
S8	44	'IPAC PROTEIN'
S9	427	S1 OR S2 OR S3 OR S4 OR S5 OR S8
S10	1943	S6 AND (SHIG? OR ESCHER? OR SALMON? OR YERSIN? OR RICKETT? OR BRUCELL? OR ERHLICH? OR CAMPYLOBACT? OR LEGIONELL? OR NEIS- SER? OR EDWARDSI?)/TI
S11	12678	LIPOPOLYSAC?/TI OR LPS?/TI
S12	965	S10 AND S11
S13	155	S9/1998:2004
S14	322	S12/1998:2004
S15	272	S9 NOT S13
S16	643	S12 NOT S14
S17	0	S15 AND (GRAM NEAR5 NEGATIV?)
S18	7	S15 AND (GRAM (5N) NEGATIV?)
S19	174	S15 AND (SHIG? OR ESCHER? OR SALMON? OR YERSIN? OR RICKETT? OR BRUCELL? OR ERHLICH? OR CAMPYLOBACT? OR LEGIONELL? OR NEI- SSER? OR EDWARDSI?)
S20	1	S19 AND S16

?t s18/9/all

18/9/1

DIALOG(R)File 155:MEDLINE(R)

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13404596 PMID: 9076742

**SopA, the outer membrane protease responsible for polar localization of IcsA in *Shigella flexneri*.**

Egile C; d'Hauteville H; Parsot C; Sansonetti P J

Unite de Pathogenie Microbienne Moleculaire, Unite 389 Institut National  
de la Sante et de la Recherche Medicale, Institut Pasteur, Paris, France.

Molecular microbiology (ENGLAND) Mar 1997, 23 (5) p1063-73, ISSN  
0950-382X Journal Code: 8712028

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

The spreading ability of *Shigella flexneri*, a facultative intracellular  
**Gram - negative** bacterium, within the host-cell cytoplasm is the result  
of directional assembly and accumulation of actin filaments at one pole of  
the bacterium. IcsA/ **VirG**, the 120 kDa outer membrane protein that is

required for intracellular motility, is located at the pole of the bacterium where actin polymerization occurs. Bacteria growing in laboratory media and within infected cells release a certain proportion of the surface-exposed IcsA after proteolytic cleavage. In this study, we report the characterization of the *sopA* gene which is located on the virulence plasmid and encodes the protein responsible for the cleavage of IcsA. The deduced amino acid sequence of SopA exhibits 60% identity with those of the OmpT and OmpP outer membrane proteases of *Escherichia coli*. The construction and phenotypic characterization of a *sopA* mutant demonstrated that SopA is required for exclusive polar localization of IcsA on the bacterial surface and proper expression of the motility phenotype in infected cells.

Tags: Human; Support, Non-U.S. Gov't

Descriptors: \*Bacterial Proteins--genetics--GE; \*Bacterial Proteins--metabolism--ME; \*DNA-Binding Proteins--genetics--GE; \*DNA-Binding Proteins--metabolism--ME; \*Dysentery, Bacillary--genetics--GE; \*Shigella flexneri--genetics--GE; \*Transcription Factors--genetics--GE; \*Transcription Factors--metabolism--ME; Actins--immunology--IM; Actins--metabolism--ME; Amino Acid Sequence; Bacterial Outer Membrane Proteins--genetics--GE; Bacterial Proteins--analysis--AN; Bacterial Proteins--immunology--IM; Blotting, Southern; Cells, Cultured; Cloning, Molecular; DNA-Binding Proteins--immunology--IM; *Escherichia coli*--genetics--GE; Fluorescent Antibody Technique, Direct; Gene Expression Regulation, Bacterial; Hela Cells; Molecular Sequence Data; Plasmids; Polymerase Chain Reaction; Recombination, Genetic; Transcription Factors--immunology--IM; Transcription, Genetic; Virulence--genetics--GE

Molecular Sequence Databank No.: GENBANK/U73461

CAS Registry No.: 0 (Actins); 0 (Bacterial Outer Membrane Proteins); 0 (Bacterial Proteins); 0 (DNA-Binding Proteins); 0 (Plasmids); 0 (Transcription Factors); 0 (virG protein); 134632-13-6 (OmpX protein)

Enzyme No.: EC 3.4.- (SopA protein)

Record Date Created: 19970617

Record Date Completed: 19970617

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DIALOG(R) File 155:MEDLINE(R)

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12681451 PMID: 7604035

**Shigella flexneri surface protein IcsA is sufficient to direct actin-based motility.**

Goldberg M B; Theriot J A

Department of Microbiology and Immunology, Albert Einstein College of Medicine, Bronx, NY 10461, USA.

Proceedings of the National Academy of Sciences of the United States of America (UNITED STATES) Jul 3 1995, 92 (14) p6572-6, ISSN 0027-8424

Journal Code: 7505876

Contract/Grant No.: AI35817; AI; NIAID

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

*Shigella flexneri* is a Gram - negative bacterial pathogen that can grow directly in the cytoplasm of infected host cells and uses a form of actin-based motility for intra- and intercellular spread. Moving intracellular bacteria are associated with a polarized "comet tail" composed of actin filaments. IcsA, a 120-kDa outer membrane protein necessary for actin-based motility, is located at a single pole on the surface of the organism, at the junction with the actin tail. Here, we demonstrate that stable expression of IcsA on the surface of *Escherichia coli* is sufficient to allow actin-dependent movement of *E. coli* in cytoplasmic extracts, at rates comparable to the movement of *S. flexneri* in infected cells. Thus, IcsA is the sole *Shigella*-specific factor required for actin-based motility. Continuous protein synthesis and polarized distribution of the protein are not necessary for actin tail formation or

movement. *Listeria monocytogenes* is an unrelated bacterial pathogen that exhibits similar actin-based intracytoplasmic motility. Actin filament dynamics in the comet tails associated with the two different organisms are essentially identical, which indicates that they have independently evolved mechanisms to interact with the same components of the host cytoskeleton.

Tags: Female; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Descriptors: \*Actins--physiology--PH; \*DNA-Binding Proteins--physiology--PH; \*Shigella flexneri--physiology--PH; \*Transcription Factors--physiology--PH; Animals; Bacterial Proteins--physiology--PH; Cell Movement; Cloning, Molecular; DNA-Binding Proteins--biosynthesis--BI; DNA-Binding Proteins--isolation and purification--IP; Escherichia coli; Fluorescent Antibody Technique; Gene Expression; Oocytes--cytology--CY; Oocytes--physiology--PH; Plasmids; Recombinant Proteins--biosynthesis--BI; Recombinant Proteins--isolation and purification--IP; Recombinant Proteins--metabolism--ME; Shigella flexneri--cytology--CY; Shigella flexneri--genetics--GE; Transcription Factors--biosynthesis--BI; Transcription Factors--isolation and purification--IP; Xenopus laevis

CAS Registry No.: 0 (Actins); 0 (Bacterial Proteins); 0 (DNA-Binding Proteins); 0 (Plasmids); 0 (Recombinant Proteins); 0 (Transcription Factors); 0 (virG protein)

Record Date Created: 19950810

Record Date Completed: 19950810

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DIALOG(R) File 155:MEDLINE(R)

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09766565 PMID: 7687249

**DNA sequence and units of transcription of the conjugative transfer gene complex (trs) of Staphylococcus aureus plasmid pG01.**

Morton T M; Eaton D M; Johnston J L; Archer G L

Department of Microbiology and Immunology, Medical College of Virginia/Virginia Commonwealth University, Richmond 23298-0049.

Journal of bacteriology (UNITED STATES) Jul 1993, 175 (14) p4436-47, ISSN 0021-9193 Journal Code: 2985120R

Contract/Grant No.: AI GM21772; AI; NIAID

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

The conjugative transfer genes of 52-kb staphylococcal R plasmid pG01 were localized to a single BglIII restriction fragment and cloned in *Escherichia coli*. Sequence analysis of the 13,612-base transfer region, designated trs, identified 14 intact open reading frames (ORFs), 13 of which were transcribed in the same direction. Each ORF identified was preceded by a typical staphylococcal ribosomal binding sequence, and 10 of the 14 proteins predicted to be encoded by these ORFs were seen when an *E. coli* in vitro transcription-translation system was used. Functional transcription units were identified in a *Staphylococcus aureus* host by complementation of Tn917 inserts that abolished transfer and by Northern (RNA) blot analysis of pG01 mRNA transcripts. These studies identified three complementation groups (trsA through trsC, trsD through trsK, and trsL-trsM) and four mRNA transcripts (trsA through trsC [1.8 kb], trsA-trsB [1.3 kb], trsL-trsM [1.5 kb], and trsN [400 bases]). No definite mRNA transcript was seen for the largest complementation group, trsD through trsK (10 kb). Comparison of predicted trs-encoded amino acid sequences to those in the data base showed 20% identity of trsK to three related genes necessary for conjugative transfer of plasmids in **gram - negative** species and 32% identity of trsC to a gene required for conjugative mobilization of plasmid pC221 from staphylococci.

Tags: Comparative Study; Support, U.S. Gov't, P.H.S.

Descriptors: \*Conjugation, Genetic; \*DNA, Bacterial--genetics--GE; \*Genes, Bacterial; \*Multigene Family; \*Plasmids; \*Staphylococcus aureus--genetics--GE; \*Transcription, Genetic; Amino Acid Sequence; Base Sequence; Blotting, Northern; Cloning, Molecular; DNA, Bacterial--isolation and



purification--IP; Escherichia coli--genetics--GE; Genetic Complementation Test; Molecular Sequence Data; Mutagenesis, Insertional; Oligodeoxyribonucleotides; Open Reading Frames; Polymerase Chain Reaction; Promoter Regions (Genetics); RNA, Bacterial--isolation and purification--IP; RNA, Messenger--metabolism--ME; Restriction Mapping; Sequence Homology, Amino Acid; Software; Terminator Regions (Genetics); Transduction, Genetic; Transformation, Bacterial; Translation, Genetic  
Molecular Sequence Databank No.: GENBANK/L11998  
CAS Registry No.: 0 (DNA, Bacterial); 0 (Oligodeoxyribonucleotides); 0 (Plasmids); 0 (RNA, Bacterial); 0 (RNA, Messenger)  
Gene Symbol: traD; traG; trs; trsB; trsC; trsD; trsH; trsI; trsJ; trsL; trsM; virD4; **virG**  
Record Date Created: 19930813  
Record Date Completed: 19930813

18/9/4

DIALOG(R) File 155:MEDLINE(R)  
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09329794 PMID: 1602963

**Phosphorylation of IcsA by cAMP-dependent protein kinase and its effect on intracellular spread of Shigella flexneri.**

d'Hauteville H; Sansonetti P J  
Unite de Pathogenie Microbienne Moleculaire, Institut National de la Sante et de la Recherche Medicale, Institut Pasteur, Paris, France.  
Molecular microbiology (ENGLAND) Apr 1992, 6 (7) p833-41, ISSN 0950-382X Journal Code: 8712028  
Document type: Journal Article  
Languages: ENGLISH  
Main Citation Owner: NLM  
Record type: Completed  
Subfile: INDEX MEDICUS

Shigella flexneri, a **Gram - negative** bacillus belonging to the family Enterobacteriaceae, causes bacillary dysentery in humans by invading colonic epithelial cells. Processes by which epithelial cells, which are not professional phagocytes, may limit the spread of the invading microorganisms are poorly understood. This paper shows that IcsA (**VirG**), a 120 kDa bacterial outer membrane protein responsible for intracellular and cell-to-cell spread through polymerization of actin, is a major substrate for phosphorylation by cyclic-dependent protein kinases. Site-directed mutagenesis of a sequence encoding phosphorylation consensus motif SSRRASS, located at residues 754-760, almost completely abolished the ability of this protein to be phosphorylated by protein kinase A. Such mutants expressed a 'super lcs' phenotype, characterized by an increased capacity to spread from cell-to-cell during the first three hours of infection in the HeLa cell infection assay. These data suggest that host-cell phosphorylation of key virulence proteins located on the bacterial surface may represent a significant host defence mechanism during the invasion process.

Tags: Human; Support, Non-U.S. Gov't  
Descriptors: \*Bacterial Proteins--metabolism--ME; \*DNA-Binding Proteins; \*Protein Kinases--metabolism--ME; \*Shigella flexneri--metabolism--ME; \*Transcription Factors; Amino Acid Sequence; Bacterial Proteins--genetics--GE; Base Sequence; Cloning, Molecular; DNA, Bacterial; HeLa Cells; Molecular Sequence Data; Mutagenesis, Site-Directed; Phenotype; Phosphorylation; Restriction Mapping; Shigella flexneri--pathogenicity--PY; Substrate Specificity; Virulence  
CAS Registry No.: 0 (Bacterial Proteins); 0 (DNA, Bacterial); 0 (DNA-Binding Proteins); 0 (Transcription Factors); 0 (virG protein).  
Enzyme No.: EC 2.7.1.37 (Protein Kinases)  
Record Date Created: 19920710  
Record Date Completed: 19920710

18/9/5

DIALOG(R) File 155:MEDLINE(R)

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09244147 PMID: 1554236

**[Molecular and cellular bases of the virulence of Shigella flexneri]**

Bases moléculaires et cellulaires de la virulence de Shigella flexneri.

Sansonetti P J

Unité de pathogénie microbienne moléculaire, Unité INSERM U199, Institut Pasteur, Paris.

Annales de gastroenterologie et d'hépatologie (FRANCE) Jan-Feb 1992,  
28 (1) p44-7, ISSN 0066-2070 Journal Code: 0263111

Document type: Journal Article ; English Abstract

Languages: FRENCH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Shigella flexneri, a Gram negative bacillus, causes bacillary dysentery, an ulcerative disease of the human colon, by invading intestinal epithelial cells. Entry into epithelial cells occurs via an induced phagocytic process which involves the actino-myosin complex. The host-cell receptor and the transmembrane signal which initiate reorganization of the cytoskeleton are under study. Binding to integrins has recently been demonstrated in related models such as the entry of Yersinia pseudotuberculosis and Bordetella pertussis into cells. Bacterial genes necessary to achieve entry are located on five contiguous loci covering 30 kb on a 220 kb virulence plasmid in S. flexneri. Locus 2 has been particularly studied. Six genes organized as an operon encode highly immunogenic proteins among which IpaB (62 kD) and IpaC (48 kD) are the invasins of this microorganism which subsequently grows very rapidly within infected cells due to its capacity to lyse the membrane bound phagocytic vacuole. Once free within the cytoplasm, bacteria interact again with the cell cytoskeleton. They first express Olm (organelle like movement), a phenotype reflecting intracellular movement along actin stress cables. They subsequently express lcs (intracellular spread), a phenotype by which intracellular bacteria induce nucleation and polymerization of actin followed by accumulation of this material at one end of the bacillus. This process causes rapid random movement leading to the formation of protrusions which allow passage to adjacent cells. A combination of these two movements achieves bacterial colonization of the epithelium.

Tags: Comparative Study

Descriptors: \*Shigella flexneri--pathogenicity--PY; Chromosome Mapping; Cytoplasm--microbiology--MI; Cytoskeleton--microbiology--MI; Endocytosis; Epithelial Cells; Epithelium--microbiology--MI; Genes, Bacterial; Intestines--cytology--CY; Intestines--microbiology--MI; Phagocytosis; Plasmids; Shigella flexneri--genetics--GE; Shigella flexneri--growth and development--GD; Virulence

CAS Registry No.: 0 (Plasmids)

Record Date Created: 19920430

Record Date Completed: 19920430

18/9/6

DIALOG(R) File 155:MEDLINE(R)

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09114346 PMID: 1742620

**[Molecular and cellular bases of Shigella flexneri virulence]**

Bases moléculaires et cellulaires de la virulence de Shigella flexneri.

Sansonetti P J

Unité de pathogénie microbienne moléculaire, Unité INSERM U199, Institut Pasteur, Paris, France.

Bulletin de l'Académie nationale de médecine (FRANCE) Jun-Jul 1991,  
175 (6) p803-9; discussion 809-10, ISSN 0001-4079 Journal Code:  
7503383

Document type: Journal Article ; English Abstract

Languages: FRENCH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

*Shigella flexneri*, a **Gram negative** bacillus, causes bacillary dysentery, an ulcerative disease of the human colon, by invading intestinal epithelial cells. Entry into epithelial cells occurs via an induced phagocytic process which involves the actino-myosin complex. The host-cell receptor and the transmembrane signal which initiate reorganization of the cytoskeleton are under study. Binding to integrins has recently been demonstrated in related models such as the entry of *Yersinia pseudotuberculosis* and *Bordetella pertussis* into cells. Bacterial genes necessary to achieve entry are located on five contiguous loci covering 30 kb on a 220 kb virulence plasmid in *S. flexneri*. Locus 2 has been particularly studied. Six genes organized as an operon encode highly immunogenic proteins among which **IpaB** (62 kD) and **IpaC** (48 kD) are the invasins of this microorganism which subsequently grows very rapidly within infected cells due to its capacity to lyse the membrane bound phagocytic vacuole. Once free within the cytoplasm, bacteria interact again with the cell cytoskeleton. They first express *Olm* (organelle like movement), a phenotype reflecting intracellular movement along actin stress cables. They subsequently express *Ics* (intracellular spread), a phenotype by which intracellular bacteria induce nucleation and polymerization of actin followed by accumulation of this material at one end of the bacillus. This process causes rapid random movement leading to the formation of protusions which allow passage to adjacent cells. A combination of these two movements achieves bacterial colonization of the epithelium.

Descriptors: \*Genes, Bacterial--genetics--GE; \*Phagocytosis; \**Shigella flexneri*--genetics--GE; Actins--physiology--PH; Cell Movement--genetics--GE; Cell Movement--physiology--PH; Gene Expression Regulation, Bacterial--genetics--GE; Organelles; Plasmids--genetics--GE; *Shigella flexneri*--classification--CL; *Shigella flexneri*--pathogenicity--PY

CAS Registry No.: 0 (Actins); 0 (Plasmids)

Record Date Created: 19920116

Record Date Completed: 19920116

18/9/7

DIALOG(R) File 155:MEDLINE(R)

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07693912 PMID: 2830310

**Development and testing of invasion-associated DNA probes for detection of *Shigella* spp. and enteroinvasive *Escherichia coli*.**

Venkatesan M; Buysse J M; Vandendries E; Kopecko D J

Department of Bacterial Immunology, Walter Reed Army Institute of Research, Washington, D.C. 20307-5100.

Journal of clinical microbiology (UNITED STATES) Feb 1988, 26 (2) p261-6, ISSN 0095-1137 Journal Code: 7505564

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Genetic determinants of the invasive phenotype of *Shigella* spp. and enteroinvasive *Escherichia coli* (EIEC), two common agents of bacillary dysentery, are encoded on large (180- to 210 kilobase), nonconjugative plasmids. Several plasmid-encoded antigens have been implicated as important bacterial ligands that mediate the attachment and invasion of colonic epithelial cells by the bacteria. Selected invasion plasmid antigen (*ipa*) genes have recently been cloned from *Shigella flexneri* serotype 5 into the lambda *gt11* expression vector. Portions of three *ipa* genes (***ipaB***, ***ipaC***, and ***ipaD***) were tested as DNA probes for diagnostic detection of bacillary dysentery. Under stringent DNA hybridization conditions, all three DNA sequences hybridized to a single 4.6-kilobase *HindIII* fragment of the invasion plasmids of representative virulent *Shigella* spp. and EIEC strains. No hybridization was detected in isogenic, noninvasive *Shigella* mutants which had lost the invasion plasmid or had deleted the *ipa* gene region. Furthermore, these probes did not react with over 300 other enteric and nonenteric **gram - negative** bacteria tested, including *Salmonella*,

Yersinia, Edwardsiella, Campylobacter, Vibrio, Klebsiella, Aeromonas, Enterobacter, Rickettsia, and Citrobacter spp. and various pathogenic E. coli strains. The use of unique invasion-essential gene segments as probes for the specific detection of invasive dysentery organisms should benefit both epidemiologic and diagnostic analyses of Shigella spp. and EIEC.

Tags: Human; Support, Non-U.S. Gov't

Descriptors: \*DNA, Bacterial--genetics--GE; \*Escherichia coli--genetics--GE; \*Plasmids; \*Shigella--genetics--GE; Antigens, Bacterial--genetics--GE; Cloning, Molecular; DNA Restriction Enzymes; Deoxyribonuclease HindIII; Dysentery, Bacillary--diagnosis--DI; Escherichia coli--immunology--IM; Escherichia coli--isolation and purification--IP; Genes, Bacterial; Nucleic Acid Hybridization; Phenotype; Sequence Homology, Nucleic Acid; Shigella--immunology--IM; Shigella--isolation and purification--IP

CAS Registry No.: 0 (Antigens, Bacterial); 0 (DNA, Bacterial); 0 (Plasmids)

Enzyme No.: EC 3.1.21 (DNA Restriction Enzymes); EC 3.1.21.- (Deoxyribonuclease HindIII)

Record Date Created: 19880407

Record Date Completed: 19880407

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14sep04 17:13:04 User228206 Session D2237.3

\$4.62 1.442 DialUnits File155

\$1.68 8 Type(s) in Format 9

\$1.68 8 Types

\$6.30 Estimated cost File155

\$0.75 TELNET

\$7.05 Estimated cost this search

\$7.05 Estimated total session cost 1.442 DialUnits

### Status: Signed Off. (3 minutes)

First Hit   Fwd Refs

L1: Entry 3 of 9

File: USPT

Jan 20, 2004

US-PAT-NO: 6680374

DOCUMENT-IDENTIFIER: US 6680374 B2

TITLE: Invaplex from gram negative bacteria, method of purification and methods of use

DATE-ISSUED: January 20, 2004

## INVENTOR-INFORMATION:

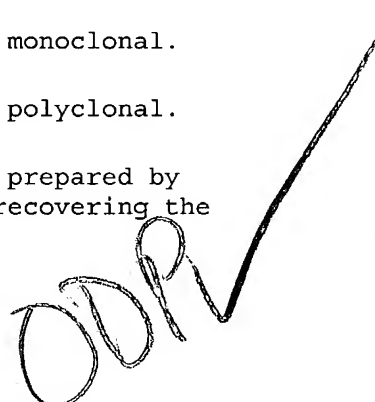
NAME	CITY	STATE	ZIP CODE	COUNTRY
Oaks; Edwin V.	Gambrills	MD		
Turbyfill; Kevin Ross	Waldorf	MD		

US-CL-CURRENT: 530/388.1; 424/130.1, 424/141.1, 424/150.1, 424/164.1, 435/329, 435/332, 435/340, 530/350, 530/388.2, 530/388.4

## CLAIMS:

What is claimed is:

1. Isolated polyclonal or monoclonal antibodies that selectively bind to an Invaplex selected from Invaplex 24 or Invaplex 50, which antibodies selectively bind to the Invaplex and not the individual components thereof.
2. The antibodies of claim 1 wherein the antibodies are monoclonal.
3. The antibodies of claim 1 wherein the antibodies are polyclonal.
4. The antibodies of claim 1 wherein the antibodies are prepared by administering Invaplex 24 or Invaplex 50 to a host and recovering the antibodies.



First Hit

L1: Entry 1 of 9

File: PGPB

Dec 26, 2002

PGPUB-DOCUMENT-NUMBER: 20020197276

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20020197276 A1

TITLE: Heterologous protection induced by immunization with invaplex vaccine

PUBLICATION-DATE: December 26, 2002

## INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Oaks, Edwin V.	Gambrills	MD	US	
Turbyfill, Kevin R.	Odenton	MD	US	

APPL-NO: 10/ 150814 [PALM]

DATE FILED: May 17, 2002

## RELATED-US-APPL-DATA:

Application is a non-provisional-of-provisional application 60/292154, filed May 18, 2001,

Application is a non-provisional-of-provisional application 60/292493, filed May 21, 2001,

INT-CL: [07] A61 K 39/116

US-CL-PUBLISHED: 424/203.1

US-CL-CURRENT: 424/203.1

REPRESENTATIVE-FIGURES: NONE

## ABSTRACT:

In this application is described a composition, Invaplex, derived from a gram negative bacteria for use in generating an immune response in a subject against one or more heterologous species or strains of gram-negative bacteria.

First Hit   Fwd Refs

L1: Entry 4 of 9

File: USPT

Aug 21, 2001

US-PAT-NO: 6277379

DOCUMENT-IDENTIFIER: US 6277379 B1

TITLE: Use of purified invaplex from gram negative bacteria as a vaccine

DATE-ISSUED: August 21, 2001

## INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Oaks; Edwin V.	Gambrills	MD		
Turbyfill; Kevin Ross	Waldorf	MD		
Hartman; Antoinette Berrong	Silver Spring	MD		

US-CL-CURRENT: 424/197.11; 424/193.1, 424/203.1, 424/234.1, 424/241.1, 424/249.1,  
424/252.1, 424/258.1, 435/975, 530/350 , 536/123.1

## CLAIMS:

What is claimed is:

1. A vaccine for providing immune protection against infection with gram negative bacteria, said vaccine comprising an isolated lipopolysaccharide-protein complex isolated from a water extract of said gram-negative bacteria in an amount effective to elicit protective antibodies in a subject to said gram-negative bacteria and a pharmaceutically acceptable carrier, wherein the complex is in its native conformation and composed of at least one invasin protein associated with LPS of said gram-negative bacteria.
2. The vaccine according to claim 1 wherein said gram-negative bacteria is selected from the group consisting of Shigella, Escherichia, Salmonella, Yersinia, Rickettsia, Brucella, Ehrlichiae, Edwardsiella, Campylobacter, Legionella and Neisseria.
3. The vaccine according to claim 1 wherein the vaccine is in a form suitable for administration by a route selected from the group consisting of oral, genital, subcutaneous, intradermal, intramuscular, intranasal, and transdermal.
4. A pharmaceutical composition comprising at least one isolated lipopolysaccharide-invasin protein complex in its native conformation isolated from a water extract of a gram-negative bacteria and a pharmaceutically acceptable excipient.
5. The composition of claim 4 wherein said composition further comprises a heterologous antigen.
6. The vaccine according to claim 1, comprising a dose containing 1 ng. to 10 mg. of said isolated lipopolysaccharide-invasin protein complex.

7. The vaccine of claim 1, comprising a dose containing from 100 ng. to 500 ug of said isolated lipopolysaccharide-invasin protein complex.
8. A kit comprising a vaccine according to claim 1 in a container with printed instructions on or accompanying the container concerning the administration of the composition to a patient to protect against or treat conditions caused by a gram-negative bacterial infection.
9. A method comprising administering to a subject a vaccine comprising a pharmaceutically acceptable excipient and an effective immunizing amount of isolated lipopolysaccharide-invasin protein complex isolated from a water extract in its native conformation, for prophylactic or therapeutic use in generating an immune response in a subject with a gram-negative bacterial infection.



First Hit   Fwd Refs

L1: Entry 5 of 9

File: USPT

Jun 12, 2001

US-PAT-NO: 6245892

DOCUMENT-IDENTIFIER: US 6245892 B1

TITLE: Invaplex from gram negative bacteria, method of purification and methods of use

DATE-ISSUED: June 12, 2001

## INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Oaks; Edwin V.	Gambrills	MD		
Turbyfill; Kevin Ross	Waldorf	MD		

US-CL-CURRENT: 530/350; 424/282.1, 435/7.2, 530/416

## CLAIMS:

What is claimed is:

1. An immunogenic composition comprising an isolated lipopolysaccharide-protein complex (Invaplex) isolated from a water extract of gram-negative bacteria, wherein the complex is composed of at least one invasin protein associated with LPS of said gram-negative bacteria.

2. A composition according to claim 1 wherein said gram-negative bacteria is selected from the group consisting of Shigella, Escherichia, Salmonella, Yersinia, Rickettsia, Brucella, Ehrlichiae, Edwardsiella, Campylobacter, Legionella and Neisseria.

3. A composition according to claim 1 wherein said Invaplex comprises IpaA, IpaB, IpaC, IpaD and LPS.

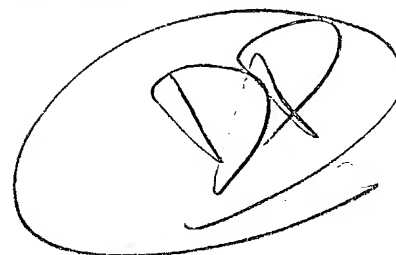
4. A composition according to claim 2 wherein said Shigella is selected from the group consisting of S. flexneri, S. sonnei, S. boydii and S. dysenteriae.

5. A composition according to claim 2 wherein said Escherichia is Escherichia coli.

6. A composition according to claim 3 wherein said Invaplex further comprises VirG or portions thereof.

7. A composition according to claim 4 wherein said Invaplex comprises at least one invasin protein selected from the group consisting of IpaA, IpaB, IpaC, IpaD and LPS.

8. A composition according to claim 5 wherein said Escherichia coli is strain EIEC.



9. A composition according to claim 8 wherein said Invaplex comprises at least one invasin protein selected from the group consisting of IpaA, IpaB, IpaC, IpaD and LPS. 9

10. A composition according to claim 8 wherein said Invaplex comprises IpaA, IpaB, IpaC, and LPS.

11. A composition according to claim 10 wherein said Invaplex further comprises IpaD.

12. A composition according to claim 11 wherein said Invaplex further comprises VirG or portions thereof.

13. A method for preparing isolated Invaplex from Shigella, said method comprising the steps of:

- (i) extracting Shigella with water to form an aqueous phase having an immunogenic lipopolysaccharide-protein complex component, Invaplex,
- (ii) separating and discarding membrane fragments from said aqueous phase resulting in a solution containing the Invaplex; and
- (iii) isolating the Invaplex from said solution.

14. The method according to claim 13 wherein said isolating in step (iii) is accomplished by using an ion exchange matrix.

15. A method for preparing isolated Invaplex from Escherichia, said method comprising the steps of:

- (i) extracting Escherichia with water to form an aqueous phase having an immunogenic lipopolysaccharide-protein complex component, Invaplex;
- (ii) separating and discarding membrane fragments from said aqueous phase resulting in a solution containing the Invaplex; and
- (iii) isolating the Invaplex from the solution with an ion-exchange matrix.

16. A method for screening agents or drugs which reduce or eliminate Invaplex said method comprising detecting a dissociation of said Invaplex in the presence of said agent or drug.

17. A method for detecting gram-negative bacterial infection in a biological sample comprising

- (i) contacting a sample with a solid surface to which is attached an Invaplex isolated from bacteria suspected of causing the bacterial infection; and
- (ii) detecting the presence or absence of a complex formed between said isolated Invaplex and antibodies specific therefor in said sample wherein the presence of said complex indicates the presence of said bacterial infection, wherein the isolated Invaplex is an immunogenic lipopolysaccharide-protein complex that has been isolated from a water extract of the gram-negative bacteria, is an adjuvant and is composed of at least one invasin protein

associated with LPS of the gram-negative bacteria.

18. The method of claim 17 wherein said biological sample is from an animal.

19. A method to elicit an antigen-specific immune response in a subject, said method comprising administering to said subject an Invaplex isolated from a gram-negative bacteria along with said antigen wherein said antigen-specific immune response is chosen from the group consisting of cell-mediated immune response, humoral immune response, and mucosal immune response, wherein the isolated Invaplex is an immunogenic lipopolysaccharide-protein complex that is isolated from a water extract of gram-negative bacteria, is an adjuvant and is composed of at least one invasin protein associated with LPS of said gram-negative bacteria.

20. The method of claim 19 wherein said antigen is selected from the group consisting of viral antigens, mammalian cell surface molecules, bacterial antigens, fungal antigens, protozoan antigens, parasitic antigens, and cancer antigens.

21. The method of claim 19 wherein said Invaplex is administered by a route selected from the group consisting of intramuscular, bronchial, genital, nasal, oral, parenteral, transcutaneous, transdermal and rectal.

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Art Unit: 1645

*Conclusion*

10. The prior art made of record and not relied upon is considered pertinent to applicant's disclosure.
11. Fasano et al (US Pat. 5,686,580; see detailed description paragraph 141) is cited to show monoclonal antibodies to IpaB and IpaC.
12. Ito et al (1991); Cortesy et al (1996); Barzu et al (1993); Pal et al (1989); Venkatesan et al (1992); and Hueck et al (1995) (abstracts) are cited to show antibodies to IpaA, IpaB, IpaC and IpaD.
13. Pace et al (US Pat. 6,083,683) is cited to show kit claims that comprise antibodies to Shigella antigens, (see claim 19).
14. Schuch et al (US Pat. 6,342,352) is cited to show antibodies to Ipa.
15. Stewart, Jr. et al (US Pat. 6,406,885) is cited to show antibodies to intimin and invasin.
16. Zychlinsky et al (US Pat. 5,972,899) is cited to show IpaB and teaches antibodies to IpaB(see detailed description paragraphs 119, 125, 127).
- 17.
18. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

13074109 PMID: 8757853

**Polysaccharide side chains are not required for attaching and effacing adhesion of Escherichia coli O157:H7.**

Cockerill F; Beebakhee G; Soni R; Sherman P

The Samuel Lunenfeld Research Institute, Mount Sinai Hospital, Toronto, Ontario, Canada.

Infection and immunity (UNITED STATES) Aug 1996, 64 (8) p3196-200,  
ISSN 0019-9567 Journal Code: 0246127

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Escherichia coli of the serotype O157:H7 is an enterohemorrhagic human pathogen which demonstrates **attaching** and **effacing** adhesion to colonocytes in vivo and to epithelial cells grown in tissue culture. Transposon TnphoA mutants of E. coli O157:H7 strain CL-8 were produced. Two of 300 alkaline phosphatase positive mutants, designated JB6 and JB27, did not express O157 side chains as assessed by agglutination with specific polyclonal O157 antiserum, silver staining of **lipopolysaccharide** extracts separated by sodium dodecyl sulfate-polyacrylamide gel electrophoresis, and Western immunoblots with polyclonal O157-specific antiserum. Both O157-negative mutants and the parent strain demonstrated localized adherence to HEp-2 cells when examined by Giemsa staining and bright-field microscopy. Furthermore, both O157-negative mutants showed enhanced adherence to HEp-2 cells compared with the parent strain when assessed by quantification of adherent bacterial CFUs. The parent strain, CL-8, and both of the mutants produced fluorescent foci when adherent bacteria and HEp-2 cells were stained with fluorescein isothiocyanate-labelled phalloidin. Transmission electron microscopy confirmed **attaching** and **effacing** adherence of strain CL-8 and the O157-negative mutants to HEp-2 cells. These findings indicate that mutants deficient in O157 polysaccharide repeats exhibit adherence to tissue culture cells in vitro and that O157 polysaccharide repeats are not required to produce the **attaching** and **effacing** lesion.

Tags: Support, Non-U.S. Gov't

Descriptors: \*Bacterial Adhesion--physiology--PH; \*Escherichia coli --physiology--PH; \*O Antigens--metabolism--ME; Agglutination Tests; Alkaline Phosphatase--genetics--GE; Bacterial Adhesion--genetics--GE; Cell Membrane--microbiology--MI; Cells, Cultured; Escherichia coli --classification--CL; Escherichia coli--genetics--GE; Mutagenesis, Insertional

CAS Registry No.: 0 (O Antigens)

Enzyme No.: EC 3.1.3.1 (Alkaline Phosphatase)

Record Date Created: 19960926

Record Date Completed: 19960926

File 155:MEDLINE(R) 1951-2004/Sep W2

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**\*File 155: Medline has been reloaded. Accession numbers**  
have changed. Please see HELP NEWS 154 for details.

Set Items Description

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?e attaching and effacing

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E2	2417	ATTACHING
E3	0	*ATTACHING AND EFFACING
E4	1	ATTACHINGS
E5	1	ATTACHMENI
E6	1	ATTACHMENOV
E7	45993	ATTACHMENT
E8	1620	ATTACHMENT //DENTURE PRECISION (DENTURE PRECISION ATTACHMENT)
E9	1154	ATTACHMENT //EPITHELIAL (EPITHELIAL ATTACHMENT)
E10	6282	ATTACHMENT //OBJECT (OBJECT ATTACHMENT)
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324 INTIMIN

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E4	35		LIPOPOLYSACCHARIDE B
E5	4		LIPOPOLYSACCHARIDE GALACTOSYLTRANSFERASE
E6	1		LIPOPOLYSACCHARIDE GLUCOSYLTRANSFERASE II
E7	1		LIPOPOLYSACCHARIDE I, YERSINIA PESTIS
E8	4		LIPOPOLYSACCHARIDE O-ANTIGEN ACETYLTRANSFERASE
E9	0	1	LIPOPOLYSACCHARIDE RECEPTORS
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S7 577 S6/1998:2004  
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841 S6  
577 S7  
S8 264 S6 NOT S7  
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264 S8  
28543 S4  
46922 S5  
S9 4 S8 AND (S4 OR S5)  
?t s9/9/all



09827851 PMID: 8376594

**Role of the *eaeA* gene in experimental enteropathogenic *Escherichia coli* infection.**

Donnenberg M S; Tacket C O; James S P; Losonsky G; Nataro J P; Wasserman S S; Kaper J B; Levine M M

Center for Vaccine Development, University of Maryland, Baltimore 21201.

Journal of clinical investigation (UNITED STATES) Sep 1993, 92 (3)

p1412-7, ISSN 0021-9738 Journal Code: 7802877

Contract/Grant No.: AI21657; AI; NIAID; AI32074; AI; NIAID; N01 AI15096; AI; NIAID

Comment in J Clin Invest. 1993 Sep;92(3) 1117-8; Comment in PMID 8376575

Document type: Clinical Trial; Journal Article; Randomized Controlled Trial

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: AIM; INDEX MEDICUS

Enteropathogenic *Escherichia coli* (EPEC) infections are a leading cause of infant diarrhea in developing countries. Recently *eaeA*, a gene necessary for the characteristic intimate attachment of EPEC to epithelial cells in tissue culture, was described. We conducted a randomized, double-blind study to determine the role of the *eaeA* gene in human EPEC infection. 11 adult volunteers ingested  $2 \times 10^{10}$  colony-forming units of O127:H6 EPEC strain E2348/69, and an equal number received the same dose of an isogenic *eaeA* deletion mutant constructed from E2348/69. Volunteers were monitored for the development of diarrhea, fever, and systemic and gastrointestinal complaints. Diarrhea developed in all 11 volunteers who received E2348/69 and in 4 of 11 who received the mutant ( $P = 0.002$ ). Fever was more common in recipients of the wild-type strain ( $P = 0.024$ ). Stool volumes were lower in recipients of the mutant. All volunteers seroconverted to E2348/69 **LPS**, but the geometric mean peak titers of serum IgG and IgA in recipients of the mutant were lower than those of recipients of the wild-type strain. IgA against **LPS** was detected in the jejunal fluid of six of six recipients of E2348/69 and 5/6 recipients of the mutant. This study unambiguously assigns a role for *eaeA* as an EPEC virulence gene, but the residual diarrhea seen in recipients of the mutant indicates that other factors are involved.

Tags: Human; Support, U.S. Gov't, P.H.S.

Descriptors: \*Adhesins, Bacterial; \*Bacterial Adhesion; \*Bacterial Outer Membrane Proteins; \*Carrier Proteins; \*Diarrhea--microbiology--MI; \*Escherichia coli--genetics--GE; \*Escherichia coli--pathogenicity--PY; \*Escherichia coli Infections--microbiology--MI; Adult; Antibodies, Bacterial--biosynthesis--BI; Double-Blind Method; Escherichia coli Infections--immunology--IM; Genes, Structural, Bacterial; Sequence Deletion  
CAS Registry No.: 0 (Adhesins, Bacterial); 0 (Antibodies, Bacterial); 0 (Bacterial Outer Membrane Proteins); 0 (Carrier Proteins); 147094-99-3 (*eae* protein)

Record Date Created: 19931015

Record Date Completed: 19931015

07783485 PMID: 3286872

**Adherence of Vero cytotoxin-producing Escherichia coli of serotype O157:H7 to human epithelial cells in tissue culture: role of outer membranes as bacterial adhesins.**

Sherman P M; Soni R

Department of Paediatrics, Hospital for Sick Children, University of Toronto, Ontario, Canada.

Journal of medical microbiology (ENGLAND) May 1988, 26 (1) p11-7,  
ISSN 0022-2615 Journal Code: 0224131

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Escherichia coli of serotype O157:H7 are Vero cytotoxin-producing enteric pathogens that have recently been associated with outbreaks of haemorrhagic colitis, sporadic cases of haemorrhagic colitis and with the haemolytic uraemic syndrome. The organisms demonstrate **attaching** and **effacing** binding to the caecum and colon of orally infected gnotobiotic piglets, chickens and infant rabbits. E. coli O157:H7 cells adhere to the surface but do not invade the cytoplasm of human epithelial cell lines in tissue culture. Since outer membranes, **lipopolysaccharides** and flagella have been identified as bacterial adhesins on other enteric pathogens, we evaluated their roles in the binding of non-fimbriated E. coli O157:H7 to HEp-2 cells. Hyperimmune rabbit antisera were prepared to whole cells, outer membranes and flagella of E. coli O157:H7. The presence of antibody to homologous antigen was confirmed by dot blot immunoassays. Both antisera and purified outer membrane and flagellar antigens were co-incubated with bacteria and HEp-2 cells to quantitate inhibition of bacterial attachment. Adherence of E. coli O157:H7 to tissue culture cells was inhibited by rabbit antisera raised to whole cells (76.0 +/- 5.6% inhibition compared with bacterial adherence in the presence of pre-immune rabbit serum) and outer membranes (69.2 +/- 3.4% inhibition). In contrast, inhibition of bacterial attachment to tissue-culture cells was significantly less when two antisera to H7 flagella were co-incubated with E. coli O157:H7 and HEp-2 cells (12.4 +/- 7.6%; 6.0 +/- 3.5% inhibition). Outer-membrane extracts inhibited adherence to E. coli O157:H7 to HEp-2 cells in a concentration dependent manner whereas isolated flagella and **lipopolysaccharide** antigens did not inhibit bacterial attachment. (ABSTRACT TRUNCATED AT 250 WORDS)

Tags: Human; Support, Non-U.S. Gov't

Descriptors: \*Bacterial Adhesion; \*Cell Membrane--physiology--PH; \*Escherichia coli--pathogenicity--PY; Bacterial Toxins--biosynthesis--BI; Cells, Cultured; Electrophoresis, Polyacrylamide Gel; Epithelium --microbiology--MI; Flagella--physiology--PH; Immune Sera; **Lipopolysaccharides** --physiology--PH; Shiga-Like Toxin I

CAS Registry No.: 0 (Bacterial Toxins); 0 (Immune Sera); 0 (Lipopolysaccharides); 0 (Shiga-Like Toxin I)

Record Date Created: 19880630

Record Date Completed: 19880630

ialog level 04.12.02D

Reconnected in file 155 15sep04 17:26:13

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File 155:MEDLINE(R) 1951-2004/Sep W2

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\*File 155: Medline has been reloaded. Accession numbers have changed. Please see HELP NEWS 154 for details.

Set Items Description

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Cost is in DialUnits

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Set	Items	Description
S1	454	ATTACH? (5N) EFFAC?
S2	326	E3-E6
S3	451	'EAE PROTEIN' OR 'EAEA' OR 'EAEA PROTEIN, E COLI' OR 'EAEB'
S4	28543	'LPS'
S5	46922	LIPOPOLYSACCHAR?
S6	841	(S1 OR S2 OR S3)
S7	577	S6/1998:2004
S8	264	S6 NOT S7
S9	4	S8 AND (S4 OR S5)
S10	326	INTIMIN? OR INVASIN? IR INVAPLEX?
S11	286	S10/1998:2004
S12	40	S10 NOT S11
S13	0	S12 AND (S4 OR S5) NOT S9
S14	0	S12 AND S4
S15	0	T S12/6/ALL

?t s12/9/9 4 29 34 1 2

12/9/9

DIALOG(R) File 155:MEDLINE(R)

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13506139 PMID: 9192001

**Immunological cross reactivity of eaeA ( intimin ) from E. coli that cause attaching and effacing lesions in humans and rabbits.**

Agin T S; Wolf M K

Walter Reed Army Institute of Research, Washington, D.C., USA.

Advances in experimental medicine and biology (UNITED STATES) 1997,  
412 p103-4, ISSN 0065-2598 Journal Code: 0121103

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Tags: Human

Descriptors: \*Adhesins, Bacterial; \*Bacterial Adhesion; \*Bacterial Outer Membrane Proteins--immunology--IM; \*Carrier Proteins; \*Escherichia coli --immunology--IM; Amino Acid Sequence; Animals; Antigens, Bacterial --immunology--IM; Cross Reactions; Rabbits

CAS Registry No.: 0 (Adhesins, Bacterial); 0 (Antigens, Bacterial); 0 (Bacterial Outer Membrane Proteins); 0 (Carrier Proteins); 147094-99-3 (eae protein)

Record Date Created: 19970826

Record Date Completed: 19970826

12/9/4

DIALOG(R) File 155:MEDLINE(R)

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13638568 PMID: 9381722

**[Enterohemorrhagic Escherichia coli and hemolytic-uremic syndrome]**

Enterohamorrhagische Escherichia coli und hamolytisch-uramisches Syndrom.  
Allerberger F; Solder B; Caprioli A; Karch H  
Bundesstaatliche bakterologisch-serologische Untersuchungsanstalt,  
Innsbruck, Osterreich.

Wiener klinische Wochenschrift (AUSTRIA) Sep 19 1997, 109 (17)  
p669-77, ISSN 0043-5325 Journal Code: 21620870R

Document type: Journal Article; Review; Review, Tutorial ; English  
Abstract

Languages: GERMAN

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Enterohemorrhagic Escherichia coli (EHEC) are increasingly identified as the cause of diarrhea and hemorrhagic colitis in countries with highly developed livestock. In 5-10% of patients, full-blown hemolytic uremic syndrome (HUS) occurs as a postinfectious life-threatening complication. Up to 1996, 5 out of 39 patients (12.8%) with EHEC O157 infections in Austria developed HUS. Acute complications of HUS such as brain edema may also lead to death; one fatal outcome has been observed so far in Austrian patients. Aside from the cytotoxic Shiga toxins, other different pathogenic factors are often found in clinical EHEC isolates. These include a cytolysin termed EHEC-hemolysin and a low molecular heat-stabile enterotoxin. Furthermore, most EHEC strains express an important surface protein, *intimin*, which is important for adherence to intestinal epithelial cells. EHEC are heterogeneous in their antigenic structure (O-, H-antigens). In Austria O157:H7 and O157:H- are the dominating serogroups; in 1997 the first Austrian case of HUS due to EHEC O26:H11 was documented. Because there are no known reliable phenotypical markers for EHEC, diagnostic strategies should focus on the demonstration of Shiga toxins or Shiga toxin genes. For epidemiological purposes it is also important to attempt to isolate the causative agent. Cows and other ruminants are reservoirs for EHEC. In the Tyrol 3% of unpasteurised milk samples, up to 10% of minced beef samples, and 6% of calves yield EHEC O157. Aside from transmission via contaminated food, direct transmission from person to person also plays a major role in the chain of EHEC infection. In contrast to Italy and Bavaria, Austria has not experienced a major outbreak due to this organism so far. A nationwide surveillance system of HUS has shown an incidence of 0.37 HUS cases per 100,000 residents in the age group 0-14 years for 1995 (Italy: 0.2 cases per 100,000; Bavaria: approx. 1.5 cases per 100,000). (73 Refs.)

Tags: Human

Descriptors: \*Escherichia coli Infections--microbiology--MI; \*Escherichia coli O157--pathogenicity--PY; \*Hemolytic-Uremic Syndrome--microbiology--MI ; Animals; Austria; Cattle--microbiology--MI; Escherichia coli Infections--diagnosis--DI; Escherichia coli Infections--transmission--TM; Food Microbiology; Hemolytic-Uremic Syndrome--diagnosis--DI; Meat--microbiology--MI; Risk Factors; Virulence

Record Date Created: 19971125

Record Date Completed: 19971125

12/9/29

DIALOG(R) File 155:MEDLINE(R)

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12934464 PMID: 8641808

**Expression of attaching/effacing activity by enteropathogenic Escherichia coli depends on growth phase, temperature, and protein synthesis upon contact with epithelial cells.**

Rosenshine I; Ruschkowski S; Finlay B B

Department of Biotechnology and Molecular Genetics, Faculty of Medicine, The Hebrew University, Jerusalem, Israel.

Infection and immunity (UNITED STATES) Mar 1996, 64 (3) p966-73,  
ISSN 0019-9567 Journal Code: 0246127

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Enteropathogenic *Escherichia coli* (EPEC) induces tyrosine phosphorylation of a 90-kDa protein (Hp90) in infected epithelial cells. This in turn facilitates intimate binding of EPEC via the outer membrane protein **intimin**, effacement of host cell microvilli, cytoskeletal rearrangement, and bacterial uptake. This phenotype has been commonly referred to as attaching/effacing (A/E). The ability of EPEC to induce A/E lesions was dependent on bacterial growth phase and temperature. Early-logarithmic-phase EPEC grown at 37 degrees C elicits strong A/E activity within minutes after infection of HeLa epithelial cells. EPEC de novo protein syntheses during the first minutes of interaction with the host cell was required to elicit A/E lesions. However, once formed, bacterial viability was not needed to maintain A/E lesions. The type of growth media and partial O<sub>2</sub> pressure level do not seem to affect the ability of EPEC to cause A/E lesions. These results indicate that the A/E activity of EPEC is tightly regulated by environmental and host factors.

Tags: Human; Support, Non-U.S. Gov't

Descriptors: \*Adhesins, Bacterial; \*Bacterial Outer Membrane Proteins--physiology--PH; \*Bacterial Proteins--biosynthesis--BI; \*Carrier Proteins; \**Escherichia coli*--pathogenicity--PY; *Escherichia coli*--growth and development--GD; *Escherichia coli*--metabolism--ME; HeLa Cells; Phosphorylation; Temperature; Tyrosine--metabolism--ME

CAS Registry No.: 0 (Adhesins, Bacterial); 0 (Bacterial Outer Membrane Proteins); 0 (Bacterial Proteins); 0 (Carrier Proteins); 147094-99-3 (eae protein); 55520-40-6 (Tyrosine)

Record Date Created: 19960716

Record Date Completed: 19960716

12/9/34

DIALOG(R) File 155:MEDLINE(R)

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12684818 PMID: 7607406

**Identification of EaeA protein in the outer membrane of attaching and effacing *Escherichia coli* O45 from pigs.**

Zhu C; Harel J; Dumas F; Fairbrother J M

Groupe de Recherche sur les Maladies Infectieuses du Porc, Universite de Montreal Faculte de Medecine Veterinaire, Saint-Hyacinthe, Quebec, Canada.

FEMS microbiology letters (NETHERLANDS) Jun 15 1995, 129 (2-3) p237-42, ISSN 0378-1097 Journal Code: 7705721

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

We have previously reported that the production of attaching and effacing lesions by *Escherichia coli* O45 isolates from pigs is associated with the eaeA (*E. coli* attaching and effacing) gene. In the present study, expression of the EaeA protein, the eaeA gene product, among swine O45 *E. coli* isolates was examined. The majority (20/22) of attaching and effacing positive, eaeA+ *E. coli* O45 isolates, but none of ten attaching and effacing negative, eaeA- or eaeA+ isolates, expressed a 97-kDa outer membrane protein as revealed by sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) and Western blot analysis. Amino-terminal amino acid sequencing demonstrated a high homology between this 97-kDa protein of swine *E. coli* O45 and the EaeA protein (**intimin**) of human enteropathogenic *E. coli* and enterohemorrhagic *E. coli*. In addition, a serological relationship between the EaeA proteins of swine O45, rabbit (RDEC-1) and human (E2348/69) attaching and effacing *E. coli* strains was observed. Our results indicate an association between expression of the EaeA protein and attaching and effacing activity among O45 *E. coli* isolates. The data also suggest an antigenic relatedness of the EaeA proteins of swine, rabbit, and human attaching and effacing *E. coli*.

Tags: Support, Non-U.S. Gov't

Descriptors: \*Adhesins, Bacterial; \*Bacterial Outer Membrane Proteins--biosynthesis--BI; \*Carrier Proteins; \**Escherichia coli*--physiology--PH;

Amino Acid Sequence; Animals; Bacterial Outer Membrane Proteins--chemistry  
--CH; Cell Adhesion; Molecular Sequence Data; Sequence Alignment; Swine  
--microbiology--MI

CAS Registry No.: 0 (Adhesins, Bacterial); 0 (Bacterial Outer Membrane  
Proteins); 0 (Carrier Proteins); 147094-99-3 (eae protein)

Record Date Created: 19950816

Record Date Completed: 19950816

12/9/1

DIALOG(R) File 155:MEDLINE(R)

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13695894 PMID: 9390560

**Enteropathogenic E. coli (EPEC) transfers its receptor for intimate  
adherence into mammalian cells.**

Kenny B; DeVinney R; Stein M; Reinscheid D J; Frey E A; Finlay B B

Department of Biochemistry and Molecular Biology, University of British  
Columbia, Vancouver, Canada.

Cell (UNITED STATES) Nov 14 1997, 91 (4) p511-20, ISSN 0092-8674

Journal Code: 0413066

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Enteropathogenic E. coli (EPEC) belongs to a group of bacterial pathogens  
that induce epithelial cell actin rearrangements resulting in pedestal  
formation beneath adherent bacteria. This requires the secretion of  
specific virulence proteins needed for signal transduction and intimate  
adherence. EPEC interaction induces tyrosine phosphorylation of a protein  
in the host membrane, Hp90, which is the receptor for the EPEC outer  
membrane protein, **intimin**. Hp90- **intimin** interaction is essential for  
intimate attachment and pedestal formation. Here, we demonstrate that Hp90  
is actually a bacterial protein (Tir). Thus, this bacterial pathogen  
inserts its own receptor into mammalian cell surfaces, to which it then  
adheres to trigger additional host signaling events and actin nucleation.  
It is also tyrosine-phosphorylated upon transfer into the host cell.

Tags: Human; Support, Non-U.S. Gov't

Descriptors: \*Adhesins, Bacterial; \*Bacterial Adhesion--genetics--GE;  
\*Bacterial Outer Membrane Proteins--metabolism--ME; \*Bacterial Proteins  
--metabolism--ME; \*Carrier Proteins; \*Escherichia coli--pathogenicity--PY;  
\*Escherichia coli Proteins; \*Receptors, Cell Surface--metabolism--ME; Amino  
Acid Sequence; Antibodies, Bacterial; Bacterial Outer Membrane Proteins  
--genetics--GE; Bacterial Outer Membrane Proteins--physiology--PH;  
Bacterial Proteins--chemistry--CH; Bacterial Proteins--genetics--GE;  
Bacterial Proteins--isolation and purification--IP; Bacterial Proteins  
--physiology--PH; Base Sequence; Cell Membrane--chemistry--CH; Cell  
Membrane--metabolism--ME; Escherichia coli--genetics--GE; Escherichia coli  
--immunology--IM; Genes, Structural, Bacterial--genetics--GE; Hela Cells;  
Isoelectric Point; Molecular Sequence Data; Molecular Weight; Mutation;  
Phosphorylation; Receptors, Cell Surface--chemistry--CH; Receptors, Cell  
Surface--genetics--GE; Receptors, Cell Surface--isolation and purification  
--IP; Recombinant Fusion Proteins--analysis--AN; Restriction Mapping;  
Tyrosine--metabolism--ME; Virulence

Molecular Sequence Databank No.: GENBANK/AF013122

CAS Registry No.: 0 (Adhesins, Bacterial); 0 (Antibodies, Bacterial);  
0 (Bacterial Outer Membrane Proteins); 0 (Bacterial Proteins); 0  
(Carrier Proteins); 0 (EaeB protein); 0 (Escherichia coli Proteins); 0  
(EspA protein); 0 (Receptors, Cell Surface); 0 (Recombinant Fusion  
Proteins); 0 (Tir protein, E coli); 147094-99-3 (eae protein);  
55520-40-6 (Tyrosine)

Record Date Created: 19971223

Record Date Completed: 19971223

12/9/2

DIALOG(R) File 155:MEDLINE(R)  
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13693500 PMID: 9387224

**Flagellar flhA, flhB and flhE genes, organized in an operon, cluster upstream from the inv locus in Yersinia enterocolitica.**

Fauconnier A; Allaoui A; Campos A; Van Elsen A; Cornelis G R; Bollen A  
Universite Libre de Bruxelles, Belgium. afaucon@sga.ulb.ac.be  
Microbiology (Reading, England) (ENGLAND) Nov 1997, 143 ( Pt 11)  
p3461-71, ISSN 1350-0872 Journal Code: 9430468

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

The inv gene of Yersinia enterocolitica codes for invasins, a member of the invasins/ intimin -like protein family, which mediates the internalization of the bacterium into cultured epithelial cells. The putative inclusion of inv into a pathogenicity island was tested by investigating its flanking sequences. Indeed, the enteropathogenic Escherichia coli (EPEC) intimin, a member of the same family of proteins, is encoded by eaeA, a gene which belongs to a pathogenicity island. An ORF located upstream from inv was of particular interest since it appeared homologous both to the flagellar flhA gene and to sepA, an EPEC gene lying inside the same pathogenicity island as eaeA. A mutant in this ORF was non-motile and non-flagellated while its invasion phenotype remained unaffected. These data indicated that the ORF corresponded to the flhA gene of Y. enterocolitica. Subsequently, the flhB and flhE genes, located respectively upstream and downstream from flhA, were identified. The three flh genes appear to be transcribed from a single operon called flhB, according to the nomenclature used for Salmonella typhimurium. Intergenic sequence between flhE and inv includes a grey hole, with no recognizable function. Downstream from inv, we have detected the flagellar flgM operon as already reported. Finally, the incongruous localization of inv amidst the flagellar cluster is discussed; while transposition could explain this phenomenon, no trace of such an event was detected.

Tags: Support, Non-U.S. Gov't

Descriptors: \*Adhesins, Bacterial; \*Bacterial Proteins--genetics--GE; \*Carrier Proteins; \*Flagella--genetics--GE; \*Membrane Proteins--genetics--GE; \*Operon--genetics--GE; \*Yersinia enterocolitica--genetics--GE; Bacterial Outer Membrane Proteins--genetics--GE; Cloning, Molecular; Escherichia coli--genetics--GE; Escherichia coli--pathogenicity--PY; Gene Expression Regulation, Bacterial--physiology--PH; Genes, Structural, Bacterial--genetics--GE; Molecular Sequence Data; Open Reading Frames--genetics--GE; RNA, Bacterial; RNA, Messenger--analysis--AN; Restriction Mapping; Sequence Analysis, DNA; Sequence Homology, Amino Acid; Temperature; Yersinia enterocolitica--pathogenicity--PY; Yersinia enterocolitica--ultrastructure--UL

Molecular Sequence Databank No.: GENBANK/Z48169

CAS Registry No.: 0 (Adhesins, Bacterial); 0 (Bacterial Outer Membrane Proteins); 0 (Bacterial Proteins); 0 (Carrier Proteins); 0 (FlhA protein); 0 (FlhB protein); 0 (FlhE protein, Salmonella typhimurium); 0 (Membrane Proteins); 0 (RNA, Bacterial); 0 (RNA, Messenger); 114073-91-5 (invasin); 147094-99-3 (eae protein)

Record Date Created: 19980210

Record Date Completed: 19980210

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15sep04 17:26:16 User228206 Session D2240.4

\$0.72 0.225 DialUnits File155

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\$1.98 Estimated cost File155

\$0.24 TELNET

\$2.22 Estimated cost this search

\$2.22 Estimated total session cost 0.225 DialUnits

### Status: Signed Off. (1 minutes)



13506139 PMID: 9192001

**Immunological cross reactivity of eaeA ( intimin ) from E. coli that cause attaching and effacing lesions in humans and rabbits.**

Agin T S; Wolf M K

Walter Reed Army Institute of Research, Washington, D.C., USA.

Advances in experimental medicine and biology (UNITED STATES) 1997,  
412 p103-4, ISSN 0065-2598 Journal Code: 0121103

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Tags: Human

Descriptors: \*Adhesins, Bacterial; \*Bacterial Adhesion; \*Bacterial Outer  
Membrane Proteins--immunology--IM; \*Carrier Proteins; \*Escherichia coli  
--immunology--IM; Amino Acid Sequence; Animals; Antigens, Bacterial  
--immunology--IM; Cross Reactions; Rabbits

CAS Registry No.: 0 (Adhesins, Bacterial); 0 (Antigens, Bacterial); 0  
(Bacterial Outer Membrane Proteins); 0 (Carrier Proteins); 147094-99-3  
(eae protein)

Record Date Created: 19970826

Record Date Completed: 19970826